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When vertigo is a stroke: Recognising posterior circulation Ischaemia in the acutely dizzy patient

Abstract

Background: Acute vertigo and dizziness are among the most common reasons for emergency presentation, and the overwhelming majority have a benign peripheral cause. A small but dangerous minority, however, are the first manifestation of posterior circulation ischaemia. These strokes are missed at first contact far more often than anterior circulation events, because isolated vertigo can be the sole symptom, the neurological examination may appear normal, and clinicians instinctively reach for imaging that performs poorly in exactly this setting.

Objective: To synthesise the contemporary evidence on how posterior circulation stroke masquerades as a peripheral vestibular disorder, and to set out a structured, examination-led approach that outperforms early neuroimaging at the bedside.

Discussion: A focused oculomotor assessment — the Head Impulse, Nystagmus, Test of Skew (HINTS) battery, extended to HINTS-plus by the addition of new unilateral hearing loss — discriminates central from peripheral acute vestibular syndrome with greater sensitivity than diffusion-weighted MRI obtained within the first 24 to 48 hours. The single most counterintuitive lesson is that a *normal* horizontal head impulse response in a patient with continuous spontaneous vertigo points towards, not away from, a stroke. The principal barriers to applying this knowledge are not gaps in the evidence but gaps in delivery: examiner training, consistent technique, and equitable access to confirmatory tools across district general and regional hospitals.

Conclusion: The acutely dizzy patient is a problem of clinical reasoning before it is a problem of technology. Disciplined bedside assessment, an honest understanding of the limits of early MRI, and investment in training and quantitative video-oculography together offer the most realistic route to closing a persistent and harmful diagnostic gap.

Keywords: acute vestibular syndrome, posterior circulation stroke, HINTS, vertigo, diffusion-weighted imaging, video-oculography, diagnostic error

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Introduction

Few presentations expose the gap between a normal-looking patient and a catastrophic diagnosis as sharply as acute dizziness. Vertigo accounts for roughly four per cent of emergency department visits, and most of those patients are sent home, correctly, with a peripheral vestibular disorder.[1] The difficulty is the residue: the patient whose only complaint is that the room is spinning, whose limbs are strong, whose speech is clear, and whose brainstem is nonetheless infarcting. Posterior circulation stroke can hide inside an entirely ordinary-seeming vestibular syndrome, and when it does, the usual reflexes of acute care — a quick neurological screen, a CT head, perhaps an early MRI — are precisely the tools most likely to give false reassurance.

This review concerns that residue. It is written for clinicians who meet the dizzy patient first — in the emergency department, the acute medical unit, and the district general hospital — and who carry the burden of deciding, often without a specialist nearby, which spinning room is a labyrinth and which is a stroke. The argument throughout is deliberately practical, and at times uncomfortable: the bedside examination, properly performed, is more reliable than the scanner in the first hours, and the reason patients are missed is rarely that the science is uncertain.

The scale of the problem

Among patients presenting to the emergency department with dizziness, vertigo or imbalance, a meaningful proportion harbour a cerebrovascular cause, and the dizzy stroke patient is disproportionately likely to be sent away unrecognised.[2] Frontline misdiagnosis of posterior circulation stroke presenting with dizziness is common, estimated in some series at around a third of cases.[3] More broadly, cerebrovascular events are missed at initial emergency presentation in a sizeable minority of patients, and the risk of misdiagnosis rises steeply when the presenting complaint is mild, transient or nonspecific — a description that fits isolated vertigo almost perfectly.[4]

The clinical cost is asymmetric. A patient with vestibular neuritis who is admitted overnight for observation loses little. A patient with a cerebellar or brainstem infarct who is discharged may return with malignant oedema, hydrocephalus, basilar thrombosis or a completed locked-in syndrome. The diagnostic task is therefore not to achieve elegance but to avoid a small number of devastating errors, and the tools must be judged against that standard.

Why the posterior fossa hides: vascular anatomy and stroke syndromes

Three vessels supply most of the territory relevant here: the posterior inferior cerebellar artery (PICA), the anterior inferior cerebellar artery (AICA), and the superior cerebellar artery. PICA-territory infarction of the inferior cerebellum or lateral medulla is the classic mimic, because a small lesion of the cerebellar nodulus or the vestibular nuclei can produce vertigo, nystagmus and imbalance with no long-tract signs whatsoever. The patient looks, to a screening examination, entirely intact.[6]

AICA-territory infarction deserves separate emphasis. The labyrinthine artery, which supplies the inner ear, most often arises from AICA, so an AICA infarct can damage the cochlea and labyrinth alongside the brainstem and cerebellum. The result is a combined audio vestibular loss — acute vertigo accompanied by new unilateral hearing impairment — that can masquerade convincingly as a peripheral problem precisely because it begins in the ear.[7] This anatomical detail is the reason hearing has earned a formal place in bedside risk stratification, discussed below. The general lesson is that the posterior fossa is unforgiving terrain: lesions are small, eloquent, and easily invisible to a clinician looking for hemiparesis or dysphasia that will never come.

Peripheral or central? The decisive bedside distinction

The relevant clinical category is the acute vestibular syndrome (AVS): the rapid onset of continuous vertigo, nausea or vomiting, nystagmus, head-motion intolerance and unsteady gait, persisting over hours to days. Most AVS is vestibular neuritis. The clinician's job is to find the minority in whom it is a stroke, and the instinct to do so by hunting for additional neurological signs is, on its own, inadequate — a large fraction of stroke patients in the seminal AVS cohort had either no obvious signs or only isolated truncal ataxia.[5]

The more powerful discriminator is the behaviour of the vestibulo-ocular reflex and the eyes themselves. This is where the examination earns its reputation, because the findings are counterintuitive: the pattern that looks reassuring to the untrained eye is the dangerous one, and the violently abnormal-looking peripheral case is usually the safe one.

The HINTS and HINTS-plus examination

The HINTS battery — Head Impulse, Nystagmus, Test of Skew — is a three-step oculomotor examination validated for the patient with continuous AVS and spontaneous nystagmus.[5,8] Each component is interpreted as follows. The horizontal head impulse test assesses the vestibulo-ocular reflex: a corrective saccade (an abnormal result) indicates a peripheral lesion, whereas an intact, *normal* head impulse in a patient with ongoing vertigo is the worrying finding, because it implies the vestibular periphery is working and the problem lies centrally.[5-6] Nystagmus that is purely horizontal and unidirectional suggests a peripheral cause; direction-changing nystagmus on eccentric gaze is central. A vertical ocular misalignment on alternate cover testing — skew deviation — is central until proven otherwise.

The mnemonic INFARCT captures the dangerous combination: Impulse Normal, Fast-phase Alternating, Refixation on Cover Test. Any one of these three central signs should prompt management as a stroke. In the original prospective study, this three-step examination was more sensitive than early diffusion-weighted MRI for posterior circulation stroke, with reported sensitivity approaching 100 per cent and specificity around 96 per cent in appropriately selected high-risk patients.[5] A subsequent systematic review confirmed strong diagnostic performance while underlining the central caveat: these figures derive substantially from specialist examiners, and accuracy in untrained hands is lower.[13]

HINTS-plus extends the battery by adding new-onset unilateral hearing loss, detectable at the bedside by finger rub, as a fourth central sign.[9] The rationale is anatomical: because the labyrinthine artery usually arises from AICA, acute hearing loss accompanying vertigo should raise rather than lower suspicion of stroke, reversing the traditional teaching that deafness implies a peripheral, ear-based problem.[7,9] The single most important safeguard against misuse is patient selection. HINTS and HINTS-plus apply only to continuous AVS with nystagmus; they are not designed for, and can mislead in, the patient with brief, positional or recurrent triggered symptoms.

The imaging trap: why early MRI reassures falsely

The most dangerous misconception in this field is that a negative scan excludes a stroke. Non-contrast CT is essentially blind to acute posterior fossa ischaemia and should never be used to rule it out. MRI is far superior, but its sensitivity in the first 24 to 48 hours is the crux of the problem. In the original HINTS cohort, diffusion-weighted imaging was falsely negative in a number of patients with brainstem and cerebellar infarction, with overall early sensitivity of 88 per cent falling to 72 per cent for the lateral medullary and pontine strokes that most closely imitate peripheral vertigo — lower than the bedside examination in the same patients.[5]

This is not an isolated finding. Small strokes causing severe vertigo are precisely those most prone to early diffusion-weighted false negatives, particularly when lesions are under ten millimetres and imaged within hours of onset.[12] The practical implication is stark: in a patient whose bedside examination indicates a central cause, a normal early MRI does not overturn that conclusion. It is the clinical assessment, repeated and supplemented by delayed imaging at three to seven days when the diagnosis remains in doubt that governs safe disposition — not a single early scan read as reassuring.

Beyond HINTS: timing, triggers and gait

HINTS is powerful but narrow, and it should sit within a broader framework. The timing-and-triggers approach asks first what kind of vestibular syndrome is present — continuous (acute vestibular syndrome), episodic and triggered, or episodic and spontaneous — because the right examination depends entirely on the answer.[11] Positional testing, not HINTS, is appropriate for the patient with brief vertigo triggered by head movement, where benign paroxysmal positional vertigo is the dominant and treatable diagnosis.

Gait remains an indispensable and underused discriminator. A patient who genuinely cannot stand or walk unaided, out of proportion to what mild vestibular neuritis would explain, should be regarded as having a central lesion until proven otherwise, irrespective of other findings.[14] Sever.e truncal ataxia may be the only abnormality in cerebellar infarction. Two further cautions are worth stating plainly: a younger patient with neck pain or headache preceding vertigo should raise the possibility of vertebral artery dissection, and the absence of vascular risk factors does not exclude stroke in this group.

Why the miss persists — and who pays for it

If the evidence is this clear, why are these strokes still missed? The honest answer is that the knowledge has not been distributed evenly to the point of care. The head impulse test is a skilled manoeuvre, easy to perform badly and easy to misinterpret; the protective finding is a normal physiological response, which runs against every instinct that equates normality with safety.[9] Misdiagnosis concentrates among non-specialists, at night, and in centres without immediate neurology or stroke expertise — in other words, in exactly the settings where most dizzy patients actually present.

The consequence is an equity problem as much as a clinical one. A patient with an AICA or PICA infarct who happens to present to a tertiary stroke centre with a trained examiner is far more likely

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to be correctly identified than the same patient presenting to a busy district general hospital overnight. The diagnostic gap is therefore not random; it tracks access. Any serious attempt to reduce harm must address the delivery of expertise to the front line, not merely the publication of better evidence. [3-4]

Closing the gap: training, technology and equitable access

Three levers are available. The first, and most immediately actionable, is training. Structured teaching of the timing-and-triggers framework and of correct HINTS technique measurably improves diagnostic accuracy among non-experts, and is far cheaper than indiscriminate imaging.[11] Embedding this into emergency and acute medicine induction, rather than leaving it to neurology, is the realistic route to scale.

The second is technology that brings the specialist examination to the bedside. Portable, quantitative video-oculography — sometimes described as an "ECG for the eyes" — measures vestibulo-ocular reflex gain and nystagmus objectively, removing the dependence on examiner skill that limits HINTS in practice.[10] Early device-based studies have reproduced expert-level discrimination of stroke from vestibular neuritis in small cohorts, and when coupled with telemedicine review by an offsite expert, such systems could extend reliable assessment to centres that will never have a neuro-otologist on site.[9-10] This, rather than ever-earlier MRI, is the more promising frontier.

The third lever is the intelligent use of imaging and pathways: reserving early MRI for the patients in whom it adds value, accepting its limitations in the first 48 hours, and building explicit safety-netting and delayed re-imaging into protocols so that a single negative early scan cannot close the case. The unifying theme is that the binding constraint is delivery and access, not the quality of the underlying evidence — and that investment in training and point-of-care quantification will avert more harm than any further refinement of the scanner.

Conclusion

Posterior circulation stroke remains one of the most consequential mimics in acute medicine, and the acutely dizzy patient is where it most often slips through. The evidence is settled enough to act on: a disciplined oculomotor examination outperforms early diffusion-weighted MRI, a normal head impulse in continuous vertigo is a red flag rather than a reassurance, new hearing loss raises suspicion rather than lowering it, and a negative early scan settles nothing. What remains unsettled is delivery — whether the clinician who meets the patient first has been trained to look, and equipped to confirm. Closing that gap is the work that will actually save brainstems, and it is a task of education and access far more than of imaging.

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Competing interests

Authors have declared that no competing interests exist.

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